CHOLERA

DISEASE REPORTING

In Washington

Since 1990, only 2 reports of *Vibrio cholerae* O1 or O139 have been received at DOH; both occurred in 2002. The last case of non-O1, O139 cholera occurred in 1996.

Cases are most often associated with travel as cholera is not endemic to Washington.

Purpose of reporting and surveillance

- To identify rare diseases associated with travel.
- To identify sources of transmission (e.g., contaminated water or a contaminated lot of shellfish*)* and to prevent further transmission from such sources.
- To identify human cases of *V. cholerae* to prevent transmission from such individuals.

Reporting requirements

- Health care providers: **immediately notifiable to Local Health Jurisdiction**
- Hospitals: **immediately notifiable to Local Health Jurisdiction**
- Laboratories: **immediately notifiable to Local Health Jurisdiction**, specimen submission required
- Local health jurisdictions: **immediately notifiable to DOH Communicable Disease Epidemiology: 1-877-539-4344**

CASE DEFINITION FOR SURVEILLANCE

Clinical criteria for diagnosis

An illness characterized by diarrhea and/or vomiting; severity is variable.

Laboratory criteria for diagnosis

- Isolation of toxigenic (i.e., cholera toxin-producing) *Vibrio cholerae* O1 or O139 from stool or vomitus, or
- Serologic evidence of recent infection.

Case definition

- Confirmed: a clinically compatible case that is laboratory confirmed.

  Illnesses caused by strains of V. cholerae *other than toxigenic* V. cholerae *O1 or O139 should not be reported as cases of cholera. The etiologic agent of a case of cholera should be reported as either* V. cholerae *O1 or* V. cholerae *O139.*

VIBRIO CHOLERAE SEROGROUPS O1 AND O139

1. Identification

An acute bacterial enteric disease characterized in its severe form by sudden onset, profuse painless watery stools, nausea and vomiting early in the course of illness, and, in untreated cases, rapid dehydration, acidosis, circulatory collapse, hypoglycemia in children, and renal failure. Asymptomatic infection is much more frequent than clinical illness, especially with organisms of the El Tor biotype; mild cases with only diarrhea are common, particularly among children. In severe untreated cases (cholera gravis), death may occur within a few hours, and the case-fatality rate may exceed 50%; with proper treatment, the rate is less than 1%.

Diagnosis is confirmed by isolating *Vibrio cholerae* of the serogroup O1 or O139 from feces. If laboratory facilities are not nearby or immediately available, Cary Blair transport medium can be used to transport or store a fecal or rectal swab. For clinical purposes, a quick presumptive diagnosis can be made by darkfield or phase microscopic visualization of the vibrios moving like “shooting stars,” inhibited by preservative free, serotype specific antiserum. For epidemiologic purposes, a presumptive diagnosis can be based on the demonstration of a significant rise in titer of antitoxic and vibriocidal antibodies. In nonendemic areas, isolated organisms from initial suspected cases should be confirmed by appropriate biochemical and serologic reactions and by testing the organisms for cholera toxin production or for the presence of cholera toxin genes. In epidemics, once laboratory confirmation and antibiotic sensitivity have been established, all cases need not be laboratory confirmed.

2. Infectious Agent

Vibrio cholerae serogroup O1 includes two biotypes-classical and El Tor-each of which includes organisms of Inaba, Ogawa and (rarely) Hikojima serotypes. *V. cholerae* O139 also causes typical cholera. The clinical pictures of illness caused by *V. cholerae* O1 of either biotype and *V. cholerae* O139 are similar because an almost identical enterotoxin is elaborated by these organisms. In any single epidemic, one particular type tends to be dominant; currently the El Tor biotype is predominant. In some areas of India and Bangladesh a proportion of clinical cholera is caused by *V. cholerae* O139 and some cases of *V. cholerae* O1 of classical biotype have been observed in Bangladesh during the past decade.

Vibrios that are biochemically indistinguishable but do not agglutinate in *V. cholerae* serogroup O1 antiserum (non-O1 strains, formerly known as nonagglutinable vibrios [NAGs] or noncholera vibrios [NCVs]) are now included in the species *V. cholerae*. Some strains elaborate cholera enterotoxin but most do not. Prior to 1992, non-O1 strains were recognized to cause sporadic cases and rare outbreaks of diarrheal disease, but were not associated with large epidemics.

However, in late 1992, large-scale epidemics of severe dehydrating diarrhea, typical of cholera, were reported in India and Bangladesh. The causative organism was a new serogroup of *V. cholerae* O139, which elaborates the same cholera toxin but differs from O1 strains in lipopolysaccharide (LPS) structure and in producing capsular antigen. The clinical and epidemiologic picture of illness caused by this organism is typical of cholera, and cases should be reported as cholera. The epidemic O139 strain, which possesses the virulence factors of *V. cholerae* O1 El Tor was apparently derived by a deletion in the genes that encode the O1 lipopolysaccharide antigen of an El Tor strain followed by the acquisition of a large fragment of new DNA encoding the enzymes that allow synthesis of O139 lipopolysaccharide and capsule. The reporting of nontoxinogenic *V. cholerae* O1 or of non-O1 *V. cholerae* infections, other than O139, as cholera is inaccurate and leads to confusion.

3. Worldwide Occurrence

During the 19th century, pandemic cholera spread repeatedly from the Ganges delta of India to most of the world. During the first half of the 20th century, the disease was confined largely to Asia, except for a severe epidemic in Egypt in 1947. During the latter half of the 20th century, the epidemiology of cholera has been marked by three major observations: 1) the relentless global spread of the seventh pandemic of cholera caused by *V. cholerae* O1 El Tor; 2) recognition that environmental reservoirs of cholera exist and include one along the Gulf of Mexico coast of the US; and 3) the appearance for the first time of large explosive epidemics of cholera gravis caused by *V. cholerae* organisms of a serogroup other than O1 (*V. cholerae* O139).

Since 1961, *V. cholerae* of the El Tor biotype has spread from Indonesia through most of Asia into eastern Europe. In 1970 this biotype was introduced into west Africa and spread rapidly throughout that continent to become endemic in many African countries. Epidemics occurred in the Iberian Peninsula and Italy in the 1970s. El Tor cholera returned to South America in 1991 after a century of absence, and caused explosive epidemics along the Pacific coast of Peru. From Peru it spread rapidly to neighboring countries and by 1994, approximately one million cholera cases had been recorded in Latin America. Notably, although the clinical disease was as severe as seen in other regions of the world, the overall case fatality in Latin America was kept remarkably low (about 1%) except in highly rural areas in the Andes and Amazon region where patients were often far from medical care.

A particularly explosive outbreak of El Tor cholera occurred among Rwandan refugees in Goma, Zaire in July, 1994, that resulted in approximately 70,000 cases and 12,000

deaths over the course of little more than one month. In total, 384,403 cases and 10,692 deaths were reported to WHO in 1994 by 94 countries. The global case fatality rate in 1994 was 2.8%, varying from 1% in the Americas, to 1.3% in Asia and 5% in Africa. These variations reflect differences in reporting and in access to appropriate treatment, and do not reflect alterations in virulence.

Except for two laboratory acquired cases, there was no known indigenous cholera in the Western Hemisphere between 1911 and 1973, when a case due to *V. cholerae* El Tor Inaba occurred in Texas with no known source. In 1978, and in the early 1990s there were additional sporadic *V. cholerae* O1 El Tor Inaba infections in Louisiana and Texas. The occurrence of these cases from the Gulf coast over many years, all due to a single indigenous strain led to the identification of an environmental reservoir of *V. cholerae* O1 El Tor Inaba in the Gulf of Mexico.

In October 1992, cholera outbreaks occurred simultaneously in several sites in Tamilnadu State, India. Strains isolated from these outbreaks did not agglutinate in O1 antisera nor were they typable with any of the standard panel of 138 non-O1 *V. cholerae* antisera. The new serogroup, designated O139 Bengal, spread rapidly throughout the region over the next few months affecting several hundred thousand persons. During this epidemic period, *V. cholerae* O139 almost completely replaced *V. cholerae* O1 strains in hospitalized cholera patients and in samples of surface water. The epidemic continued to spread through 1994, with cases of O139 cholera reported from 11 countries in Asia. This new strain was soon introduced into other continents by infected travelers, but secondary spread outside of Asia has not been reported. In the early 1990s it was believed that the O139 epidemics in Asia might be the beginning of an eighth pandemic of cholera. However, not only did O139 not spread to cause epidemic disease in Africa and South America, it greatly diminished in India and Bangladesh, and disappeared from areas to which it had spread and did not account for more than 5-10% of cases anywhere. Cholera O139 may in the future cause large explosive epidemics in another region of the world and therefore requires continued international surveillance.

Since cholera returned to Latin America in the early 1990s, cases of traveler's cholera have greatly increased. Moreover, by using optimized bacteriologic methods (TCBS medium) several prospective studies have demonstrated that the incidence of traveler's cholera in US and Japanese travelers is considerably higher than had been previously estimated.

4. Reservoir

Humans; observations in the US, Bangladesh and Australia over the past two decades have clearly demonstrated that environmental reservoirs exist, apparently in association with copepods or other zooplankton in brackish water or estuaries.

5. Modes of Transmission

Through ingestion of food or water contaminated directly or indirectly with feces or vomitus of infected persons. El Tor and O139 organisms can persist in water for long periods. When epidemic El Tor cholera appeared in Latin America in explosive fashion in 1991, faulty municipal water systems, contaminated surface waters, and unsafe domestic water storage methods resulted in extensive waterborne transmission of cholera. Beverages prepared with contaminated water and sold by street vendors, ice and even commercial bottled water were incriminated. Cooked grains with sauces have been incriminated as vehicles in cholera transmission. *V. cholerae* introduced by a food handler into one of these foods and stored unrefrigerated can increase by several logs within 8-12 hours. Vegetables and fruit "freshened" with untreated sewage wastewater have also served as vehicles of transmission. Outbreaks or epidemics as well as sporadic cases are often attributed to raw or undercooked seafood. Sometimes these vehicles come from polluted waters as in outbreaks on Guam, Kiribati, Portugal, Italy and Ecuador. In other instances, as in the US, sporadic cases of cholera follow the ingestion of raw or inadequately cooked seafood from nonpolluted waters. The Louisiana and Texas cases have been traced to eating shellfish from coastal and estuarine waters where a natural reservoir of *V. cholerae* O1, serotype Inaba, appears to exist in an estuarine environment not characterized by sewage contamination. Clinical cholera in endemic areas is usually confined to the lowest socioeconomic groups.

6. Incubation period

From a few hours to 5 days, usually 2-3 days.

7. Period of communicability

Presumably as long as stools are positive, usually only a few days after recovery. Occasionally the carrier state may persist for several months. Antibiotics known to be effective against the infecting strains (e.g., tetracycline against the O139 strain and most O1 strains) shorten the period of communicability. Very rarely, chronic biliary infection that lasts for years has been observed in adults associated with intermittent shedding of vibrios in the stool.

8. Susceptibility and resistance

Variable; gastric achlorhydria increases risk of illness, and breast fed infants are protected. Cholera gravis due to the El Tor biotype and O139 vibrio occurs significantly more often among persons with blood group O. Infection with either *V. cholerae* O1 or O139 results in a rise in agglutinating and antitoxic antibodies, and increased resistance to reinfection. Serum vibriocidal antibodies, which are readily detected following O1 infection (but for which comparably specific, sensitive and reliable assays are not available for O139 infection), are the best immunologic correlate of protection against O1 cholera. Field studies show that an initial clinical infection by *V. cholerae* O1 of the classical biotype confers protection against either classical or El Tor biotypes; in contrast an initial clinical

infection caused by biotype El Tor results in only a modest level of long-term protection that is limited to El Tor infections. In endemic areas, most people acquire antibodies by early adulthood. However, infection with O1 strains affords no protection against O139 infection and vice versa. In experimental challenge studies in volunteers, an initial clinical infection due to *V. cholerae* O139 conferred significant protection against diarrhea upon rechallenge with *V. cholerae* O139.

B. METHODS OF CONTROL

1. Preventive measures:

a. Educate the public regarding the importance of handwashing. Provide suitable handwashing facilities; this is particularly important for food handlers and attendants involved in the care of patients and children.
b. Dispose of human feces in a sanitary manner and maintain fly proof latrines. Stress use of sufficient toilet paper to minimize finger contamination. Under field conditions, dispose of feces by burial at a site distant and downstream from the source of drinking water.
c. Protect, purify and chlorinate public water supplies, provide safe private supplies, and avoid possible back flow connections between water and sewer systems. For individual and small group protection, and while traveling or in the field, treat water chemically or by boiling.
d. Control flies by screening, spraying with insecticides and use of insecticidal baits and traps. Control fly breeding by frequent collection and disposal of garbage, and fly control measures in latrine construction and maintenance.
e. Use scrupulous cleanliness in food preparation and handling; refrigerate as appropriate. Particular attention should be directed to the proper storage of salads and other foods served cold. These provisions apply equally to home and public eating places. If uncertain about sanitary practices, select foods that are cooked and served hot, and fruits peeled by the consumer.
f. Pasteurize or boil all milk and dairy products. Supervise the sanitary aspects of commercial milk production, storage and delivery.
g. Enforce suitable quality-control procedures in industries that prepare food and drink for human consumption. Use chlorinated water for cooling during canned food processing.
h. Active immunization with the current killed whole cell vaccine given parenterally is of little practical value in epidemic control or management of contacts to cases. These vaccines have been shown to provide partial protection (50%) of short duration (3-6 months) in highly endemic areas and do not prevent asymptomatic infection; they are not recommended. Two oral vaccines that provide significant protection for several months against cholera caused by O1 strains have become available in a number of countries: One is a single-dose live vaccine (strain CVD 103-HgR, available under the trade names Orachol in Europe and Mutacol in Canada, SSVI); the other is a nonliving vaccine consisting of inactivated vibrios plus B-subunit of the

cholera toxin, given on a 2-dose schedule (Dukoral, SBL). As of late 1999, these vaccines were not licensed in the US.

i. Measures that inhibit or otherwise compromise the movement of people, foods or other goods are not justified.

2. Control of patient, contacts and the immediate environment:

a. Report to local health authority.
b. Isolation: Hospitalization with enteric precautions is desirable for severely ill patients; strict isolation is not necessary. Less severe cases can be managed on an outpatient basis with oral rehydration and an appropriate antimicrobial agent. Crowded cholera wards can be operated without hazard to staff and visitors when effective handwashing and basic procedures of cleanliness are practiced. Fly control should be practiced.
c. Concurrent disinfection: Of feces and vomitus and of linens and articles used by patients, by heat, carbolic acid or other disinfectant. In communities with a modern and adequate sewage disposal system, feces can be discharged directly into the sewers without preliminary disinfection. Terminal cleaning.
d. Quarantine: None.
e. Management of contacts: Surveillance of persons who shared food and drink with a cholera patient for 5 days from last exposure. If there is evidence or high likelihood of secondary transmission within households, household members should be given chemoprophylaxis; in adults, tetracycline (500 mg 4 times daily) or doxycycline (a single daily dose of 300 mg) for 3 days, unless local strains are known or believed to be tetracycline resistant. Children may also be given tetracycline (50 mg/kg/day in 4 divided doses) or doxycycline (a single dose of 6 mg/kg) for 3 days; with such short courses of tetracyclines, staining of teeth is not a problem. Alternative prophylactic agents that may be useful where *V. cholerae* O1 strains are resistant to tetracycline include: furazolidone (Furoxone) (100 mg 4 times daily for adults and 1.25 mg/kg 4 times daily for children); erythromycin (pediatric dosage 40 mg/kg/day in 4 divided doses; adult dosage 250 mg 4 times daily); TMP-SMX (320 mg TMP and 1600 mg SMX twice daily for adults and 8 mg/kg TMP and 40 mg/kg SMX daily in 2 divided doses for children); or ciprofloxacin (500 mg twice daily for adults). TMP-SMX is not useful for *V. cholerae* O139 infections as these strains are resistant to this antimicrobial. Mass chemoprophylaxis of whole communities is never indicated and can lead to antibiotic resistance. Immunization of contacts is not indicated.
f. Investigation of contacts and source of infection: Investigate possibilities of infection from polluted drinking water and contaminated food. Meal companions for the 5 days prior to onset should be interviewed. A search by stool culture for unreported cases is recommended only among household members or those exposed to a possible common source in a previously uninfected area.
g. Specific treatment: These are three mainstays in the treatment of patients with cholera: 1) aggressive rehydration therapy; 2) administration of effective antibiotics; and 3) treatment of complications. Aggressive rehydration by oral and intravenous routes to repair fluid and electrolyte deficits and to replace the prodigious ongoing diarrheal losses is the cornerstone of cholera therapy. Appropriate antimicrobials are

an important adjunct to fluid therapy, as they diminish the volume and duration of purging and rapidly curtail the excretion of vibrios, thereby diminishing the chance of secondary transmission. Finally, as rehydration therapy becomes increasingly effective, patients who survive from hypovolemic shock and severe dehydration manifest certain complications such as hypoglycemia that must be recognized and treated promptly. If these basic guidelines are adhered to, case fatality, even during explosive outbreaks in developing countries can be kept to less than 1%.

In initiating prompt aggressive fluid therapy with volumes of electrolyte solution adequate to correct dehydration, acidosis and hypokalemia most patients with mild or moderate fluid loss can be treated entirely with oral rehydration using solutions that contain glucose 20 g/L (or sucrose 40 g/L or cooked rice powder 50 g/L); NaCl (3.5 g/L); KCl (1.5 g/L); and trisodium citrate dihydrate (2.9 g/L) or NaHCO3 (2.5 g/L). Mild and moderate volume depletion should be corrected with oral solutions by replacing, over 4-6 hours, a volume matching the estimated fluid loss (approximately 5% of body weight for mild and 7% for moderate dehydration). Continuing losses are replaced by giving, over 4 hours, a volume of oral solution 1.5 times the stool volume lost in the previous 4 hours.

Patients in shock should be given rapid IV rehydration with a balanced multielectrolyte solution containing approximately 130 mEq/L of Na+, 25-48 mEq/L of bicarbonate, acetate or lactate ions, and 10-15 mEq/L of K+. Useful solutions include Ringer's lactate or WHO “diarrhea treatment solution” (4 g NaCl, 1 g KCl, 6.5 g sodium acetate and 8 g glucose/L), and “Dacca solution” (5 g NaCl, 4 g NaHCO3 and 1 g KCl/L), which can be prepared locally in an emergency. The initial fluid replacement should be 30 ml/kg in the first hour for infants and in the first 30 minutes for persons over 1 year of age, after which the patient should be reassessed. After circulatory collapse has been effectively reversed, most patients can be switched to oral rehydration to complete the 10% initial fluid deficit replacement and to match continuing fluid loss.

Appropriate antimicrobial agents can shorten the duration of diarrhea, reduce the volume of rehydration solutions required, and shorten the duration of vibrio excretion. Adults are given tetracycline 500 mg 4 times a day, and children 12.5 mg/kg 4 times daily, for 3 days. When tetracycline resistant strains of *V. cholerae* are prevalent, alternative antimicrobial regimens include TMP-SMX (320 mg trimethoprim and 1600 mg sulfamethoxazole twice daily for adults and 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole daily in 2 divided doses for children, for 3 days); furazolidone (100 mg 4 times daily for adults and 1.25 mg/kg 4 times daily for children, for 3 days); or erythromycin (250 mg 4 times daily for adults and 10 mg/kg 3 times daily for children, for 3 days). Ciprofloxacin, 250 mg once daily for three days, is also a useful regimen for adults. *V. cholerae* O139 strains are resistant to TMP-SMX. Since individual strains of *V. cholerae* O1 or O139 may be resistant to any of these antimicrobials, knowledge of the sensitivity of local strains to these agents, if available, should be used to guide the choice of the antimicrobial therapy.

3. Epidemic measures

a. Educate the population at risk concerning the need to seek appropriate treatment without delay.
b. Provide effective treatment facilities.
c. Adopt emergency measures to ensure a safe water supply. Chlorinate public water supplies, even if the source water appears to be uncontaminated. Chlorinate or boil water used for drinking, cooking and washing dishes and food containers unless the water supply is adequately chlorinated and subsequently protected from contamination.
d. Ensure careful preparation and supervision of food and drinks. After cooking or boiling, protect against contamination by flies and unsanitary handling; leftover foods should be thoroughly reheated before ingestion. Persons with diarrhea should not prepare food or haul water for others. Food served at funerals of cholera victims may be particularly hazardous and should be discouraged during epidemics.
e. Initiate a thorough investigation designed to find the vehicle and circumstances (time, place, person) of transmission, and plan control measures accordingly.
f. Provide appropriate safe facilities for sewage disposal.
g. Parenteral whole cell vaccine is not recommended.
h. If local conditions are relatively settled, the new oral cholera vaccines can serve as an additional adjunct measure to aid in cholera control. However, these vaccines should not be used in chaotic situations or where there is a severe shortage of water that interferes with the provision of oral rehydration therapy.

4. International measures

a. Governments are required to report by telegraph to WHO and adjacent countries the first imported, first transferred or first nonimported case of cholera due to *V. cholerae* O1 or O139 in an area previously free of the disease. In the US, clinicians and microbiologists report suspected cases to their state epidemiologist; state health departments then notify the CDC, which confirms the case and notifies WHO.
b. Measures applicable to ships, aircraft and land transport arriving from cholera areas are specified in International Health Regulations (1969), Third Annotated Edition 1983, Updated and Reprinted 1992, WHO, Geneva.
c. International travelers: Immunization with the parenteral whole cell vaccine is not recommended by WHO for travel from country to country in any part of the world and is not officially required by any country. Immunization with either of the new oral vaccines is recommended for individuals from industrialized countries traveling to areas of endemic or epidemic cholera. In those countries where the new oral vaccines are already licensed, immunization is particularly recommended for travelers who have known risk factors such as individuals with hypochlorhydria (consequent to partial gastrectomy or medication) or cardiac disease (e.g., arrhythmias), the elderly, or any individuals of blood group O. As of late 1999, these vaccines were not licensed in the US. International Health Regulations state that ". . . a person on an international voyage, who has come from an infected area within

the incubation period of cholera and who has symptoms indicative of cholera, may be required to submit to stool examination."

d. WHO Collaborating Centres.

VIBRIO CHOLERAE SEROGROUPS OTHER THAN O1 AND O139

A. DESCRIPTION

1. Identification

Of the more than 100 *V. cholerae* serogroups that exist, only O1 and O139 are associated with the epidemiologic features and clinical syndrome of cholera. However, organisms of *V. cholerae* serogroups other than O1 and O139 have been associated with sporadic cases and small outbreaks of gastroenteritis. They have also rarely been isolated from patients with septicemic disease (usually immunocompromised hosts).

2. Infectious Agent

V. cholerae pathogens of serogroups other than O1 and O139. As with all *V. cholerae*, growth is enhanced in an environment of 1% NaCl. Rarely do non-O1/non-O139 *V. cholerae* strains elaborate cholera toxin or harbor the colonization factors of O1 and O139 epidemic strains. Some non-O1/non-O139 strains make a heat stable enterotoxin (so-called NAG-ST). Epidemiologic and volunteer challenge studies have documented the pathogenicity of strains that produce NAG-ST. The non-O1/non-O139 strains isolated from blood of septicemic patients have been heavily encapsulated.

3. Worldwide Occurrence

Non-O1/non-O139 *V. cholerae* strains are associated with 2%-3% of cases (including travelers) of diarrheal illness in tropical developing countries. Isolation rates are higher in coastal areas.

4. Reservoir

Non-O1/non-O139 *V. cholerae* are found in aquatic environments worldwide, particularly in mildly brackish waters where they constitute autochthonous flora. Although these organisms are halophilic, they can also proliferate in fresh water (e.g., lakes). *Vibrio* counts vary with season and peak in warm seasons. In brackish waters they are found adherent to chitinous zooplankton and shellfish.

5. Modes of Transmission

Cases of non-O1/non-O139 gastroenteritis are usually linked to consumption of raw or undercooked seafood, particularly shellfish. In tropical endemic areas, some infections may be due to ingestion of surface waters. Wound infections arise from environmental exposure, usually to brackish water or from occupational accidents among fishermen,

shellfish harvesters, etc. In high risk hosts septicemia may result from a wound infection or from ingestion of contaminated seafood.

6. Incubation period

Short. From 12 to 24 hours in outbreaks and an average of 10 hours in experimental challenge of volunteers (range 5.5 to 96 hours).

7. Period of communicability

It is not known whether in nature these infections can be transmitted from person to person or by humans contaminating food vehicles. If the latter indeed occurs the period of potential communicability would likely be limited to the period of vibrio excretion, usually several days.

8. Susceptibility and resistance

All humans are believed to be susceptible to gastroenteritis if they ingest a sufficient number of non-O1/non-O139 *V. cholerae* in an appropriate food vehicle or to develop a wound infection if the wound is exposed to vibrio containing water or shellfish. Septicemia develops only in abnormal hosts such as those who are immunocompromised, have chronic liver disease or severe malnutrition.

B. METHODS OF CONTROL

1. Preventive measures:

a. Educate consumers about the risks associated with eating raw seafood unless it has been irradiated.
b. Educate seafood handlers and processors on the following preventive measures:
    i. Ensure that cooked seafood reaches temperatures adequate to kill the organism by heating for 15 minutes at 70ºC/158ºF (organisms may survive at 60ºC/140ºF for up to 15 minutes and at 80ºC/176ºF for several minutes).
    ii. Handle cooked seafood in a manner that precludes contamination from raw seafood or contaminated seawater.
    iii. Keep all seafood, raw and cooked, adequately refrigerated before eating.
    iv. Avoid use of seawater in food handling areas, e.g., on cruise ships.

2. Control of patient, contacts and the immediate environment:

a. Report to local health authority.
b. Isolation: Enteric precautions.
c. Concurrent disinfection: Not pertinent.
d. Quarantine: Not pertinent.
e. Management of contacts: Not pertinent.

f. Investigation of contacts and source of infection: Not pertinent. Control is of outbreaks; single cases are rarely identified.
g. Specific treatment: Fluid replacement when indicated.

Patients with liver disease or who are immunosuppressed (by treatment or underlying disease) and alcoholics should be warned not to eat raw seafood. When disease occurs in these individuals, a history of eating seafood and especially the presence of bullous skin lesions justify early institution of antibiotic therapy, with a combination of oral minocycline (100 mg every 12 h) and intravenous cefotaxime (2 grams every 8 h) as the treatment regimen of choice. Tetracyclines and ciprofloxacin are also effective.

3. Epidemic measures

a. By quick review of reported cases, determine time and place of exposure and the population at risk; obtain a complete listing of the foods served and embargo, under refrigeration, all foods still available. The prominent clinical features, coupled with an estimate of the incubation period, provide useful leads to the most probable etiologic agent. Collect specimens of feces for laboratory examination; alert the laboratory to suspected etiologic agents. Interview a random sample of those exposed. Compare the attack rates for specific food items eaten and not eaten; the implicated food item(s) will usually have the greatest difference in attack rates. Most of the sick will have eaten the contaminated food.
b. Inquire about the origin of the incriminated food and the manner of its preparation and storage before serving. Look for possible sources of contamination and periods of inadequate refrigeration and heating that would permit growth of bacteria. Submit any leftover suspected foods promptly for laboratory examination.

4. International measures

WHO Collaborating Centres.